Tuberculous Pericarditis: Optimal Diagnosis and Management

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Pericarditis is a rare manifestation of tuberculous disease. The appropriate diagnostic workup and optimal therapeutic management are not well defined. We present 10 new cases of tuberculous pericarditis and review the relevant literature. The specific topics addressed are (1) the importance of tissue for diagnosis, (2) the optimal surgical management, (3) the role of corticosteroids, and (4) the impact of human immunodeficiency virus (HIV) on the management of this disease. The cases and the literature suggest that the optimal management includes an open pericardial window with biopsy, both for diagnosis and to prevent reaccumulation of fluid. Corticosteroids probably offer some benefit in preventing fluid reaccumulation as well. The data are inconclusive regarding whether open drainage or corticosteroid use prevents progression to constrictive pericarditis. No studies have addressed these issues specifically in HIV-positive patients, but the 3 HIV-positive patients in our series had an excellent response to drainage and antituberculous therapy.

Pericarditis is a rare manifestation of tuberculous disease that can be fatal even with proper diagnosis and treatment. Because of the rarity of this illness and the scarcity of informative studies, the appropriate diagnostic workup is not common knowledge, and the optimal management has not been well defined. Even less is known about the management of tuberculous pericarditis in HIV-infected patients. In this article, 1 patient who we treated for tuberculous pericarditis is described in detail, and 9 other cases are summarized in tabular form (table 1). These 9 cases were identified by reviewing the infectious disease consult records, the tuberculosis control records, and the codes of the International Classification of Diseases, 9th edition, on hospital charts in the Texas Medical Center for 1 January 1990 through 1 May 2000. The criteria for diagnosis were as follows: (1) culture of either pericardial tissue or fluid specimens that was positive for *Mycobacterium tuberculosis*, (2) granulomas and acid-fast bacilli (AFB) seen on histopathological examination of pericardial tissue, or (3) granulomas in the pericardial tissue and a positive culture for *M. tuberculosis* from another site.

The objectives of this case series and literature review are to (1) highlight the importance of obtaining pericardial tissue for diagnosis, (2) discuss the optimal surgical management, (3) address the potential role of corticosteroids, and (4) review the impact of HIV on the management of tuberculous pericarditis. No comprehensive review has previously addressed all of these issues.

**CASE REPORT**

A 39-year-old, HIV-infected, homeless man presented to the Houston Veterans Affairs Medical Center with a 6-week history of dry cough, shortness of breath, pleuritic chest pain, fever, and weight loss. The patient had not received any antiretroviral therapy in the 15 years since his diagnosis of HIV infection in 1985. He had no prior opportunistic infections. Four weeks before admission, he was evaluated for these symptoms, and the findings of a chest radiograph were normal (figure 1).

The patient was cachectic, with an oral temperature...
Tuberculous Pericarditis

Figure 1. Chest radiograph of a 39-year-old, HIV-infected, homeless man obtained 4 weeks prior to admission to the hospital.

Figure 2. Chest radiograph of a 39-year-old, HIV-infected, homeless man obtained at admission.

of 38.7°C (101.6°F) and a respiratory rate of 22 breaths/min. His blood pressure and pulse were, respectively, 109/63 mm Hg and 127 beats/min while supine and 71/51 mm Hg and 147 beats/min while standing. He had oral thrush and widespread lymphadenopathy. His neck veins were distended. His heart was enlarged with an irregular rhythm and a loud pericardial rub. The liver span was 14 cm. He had no peripheral edema.

The patient’s hemoglobin level was 9.4 g/dL, his WBC count was 5300 cells/mm³, and his platelet count was 262,000 cells/mm³. The findings of blood chemistries were normal. Laboratory studies revealed the following values: aspartate aminotransferase level, 65 U/L; alanine aminotransferase level, 93 U/L; lactate dehydrogenase level, 265 U/L; and alkaline phosphatase level, 130 U/L. His CD4 count was 36 cells/mm³ (17% of total lymphocytes), and the RNA virus load was 418,017 copies/mL. The chest radiograph showed a large globular heart shadow with clear lung fields (figure 2). The results of tuberculin and Candida skin tests were negative.

An echocardiogram revealed a large pericardial effusion without right ventricular collapse, and the patient underwent pericardial window placement with pericardial biopsy. The pericardial fluid had a RBC count of 4400 cells/mm³ and a WBC count of 60 cells/mm³ (50 lymphocytes and 10 neutrophils). Stains of pericardial fluid were negative for AFB. Examination of the pericardial biopsy specimen revealed granulomas. AFB smear of the tissue concentrate demonstrated organisms, and growth of M. tuberculosis in the BACTEC 13AC Mycobacteria Culture Vial (Becton Dickinson) was detected 9 days after inoculation. Cultures of pericardial fluid and sputum became positive on the 16th and 21st days, respectively.

Isoniazid, rifampin, pyrazinamide, and ethambutol were started immediately after the histologic examination of the pericardial tissue. Two days later, the patient’s daily fevers had subsided, and he felt very well. As an outpatient, he started receiving antiretroviral therapy, but unfortunately he died of lymphoma 5 months later. He had been free of cardiac symptoms for several months prior to his death.

DISCUSSION

The predominant symptoms of tuberculous pericarditis are cough, dyspnea, and chest pain. Night sweats, orthopnea, weight loss, and ankle edema are also common. As for signs, the most frequent are cardiomegaly, pericardial rub, fever, and tachycardia. Other findings may include pulsus paradoxicus, hepatomegaly, distended neck veins, pleural effusion, and distant heart tones [1–8]. Our patient manifested almost all of these symptoms and signs. Current understanding of the pathogenesis of tuberculosis suggests that proinflammatory cytokines may be responsible for the symptoms of fever, weight loss, and weakness. The cytokine most clearly implicated is TNF-α [9–11]. A small placebo-controlled study of thalidomide in 30 patients with pulmonary tuberculosis demonstrated that thalidomide, which is known to inhibit TNF production, decreased levels of this substance. The patients who received thalidomide also gained more weight than did the patients who...
Table 1. Summary of cases of tuberculous pericarditis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, years</th>
<th>Sex</th>
<th>Race</th>
<th>Exposure risk</th>
<th>Comorbid conditions</th>
<th>HIV</th>
<th>PPD</th>
<th>Initial procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39</td>
<td>M</td>
<td>White</td>
<td>Homeless, lived in shelters</td>
<td>Tobacco use</td>
<td>Pos\textsuperscript{a}</td>
<td>Anergic\textsuperscript{b}</td>
<td>Pericardial window and biopsy</td>
</tr>
<tr>
<td>2</td>
<td>22</td>
<td>M</td>
<td>Latin American</td>
<td>Parents with positive PPDs, incarcerated 2 weeks</td>
<td>—</td>
<td>Neg</td>
<td>Anergic</td>
<td>Pericardial window</td>
</tr>
<tr>
<td>3</td>
<td>54</td>
<td>M</td>
<td>African American</td>
<td>Partially treated pulmonary tuberculosis</td>
<td>Alcohol, tobacco abuse</td>
<td>Neg</td>
<td>—</td>
<td>Pericardial window and biopsy</td>
</tr>
<tr>
<td>4</td>
<td>16</td>
<td>M</td>
<td>Latin American</td>
<td>Raised in Mexico, positive PPD 4 years ago, no INH given</td>
<td>—</td>
<td>Neg</td>
<td>Pos</td>
<td>Pericardio-centesis</td>
</tr>
<tr>
<td>5</td>
<td>30</td>
<td>F</td>
<td>African American</td>
<td>From Uganda, Recent malaria</td>
<td>Pos\textsuperscript{c}</td>
<td>Pos</td>
<td>—</td>
<td>Pericardio-centesis</td>
</tr>
<tr>
<td>6</td>
<td>32</td>
<td>M</td>
<td>White</td>
<td>Roommate on directly-observed therapy</td>
<td>Alcohol abuse, injection drug use</td>
<td>Pos\textsuperscript{d}</td>
<td>Anergic</td>
<td>Pericardio-centesis</td>
</tr>
<tr>
<td>7</td>
<td>45</td>
<td>M</td>
<td>African American</td>
<td>Incarcerated</td>
<td>Alcohol, tobacco, injection drug use</td>
<td>Neg</td>
<td>Pos 13 years earlier</td>
<td>Pericardio-centesis</td>
</tr>
<tr>
<td>8</td>
<td>73</td>
<td>M</td>
<td>Latin American</td>
<td>Living in Puerto Rico</td>
<td>High blood pressure</td>
<td>—</td>
<td>Thoracic aortic aneurysm repair, pericardial fluid sent</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>82</td>
<td>M</td>
<td>African American</td>
<td>—</td>
<td>Tobacco use</td>
<td>—</td>
<td>Anergic</td>
<td>Pericardio-centesis</td>
</tr>
<tr>
<td>10</td>
<td>76</td>
<td>F</td>
<td>Latin American</td>
<td>—</td>
<td>Tobacco use</td>
<td>Neg</td>
<td>—</td>
<td>Pericardio-centesis</td>
</tr>
</tbody>
</table>

**NOTE.** AFB, acid-fast bacilli; INH, isoniazid; Neg, negative; PPD, purified protein derivative of tuberculin skin test; Pos, positive.

\(a\) CD4 cell count, 36 cells/mm\(^3\).

\(b\) No response to tuberculin or Candida antigen.

\(c\) CD4 cell count, 272 cells/mm\(^3\).

\(d\) CD4 cell count, 240 cells/mm\(^3\).

received placebo [12]. The nature of the T helper cell response, whether TH1 or TH2, may also influence the clinical manifestations of this disease [13].

Because the prevalence of tuberculous pericarditis varies widely with geographic location, the positive predictive value of the characteristic symptoms and signs varies as well. In African countries where both tuberculosis and HIV are endemic, and where microbiologic studies may not be readily available, symptoms or signs of pericarditis in an HIV-positive patient may be enough to prompt antituberculous therapy [14–16]. For example, in a study of Tanzanian patients with large pericardial effusions, 14 of 14 HIV-positive patients had tuberculous pericarditis [14]. However, numerous other infectious and noninfectious causes can have a presentation similar to that of tuberculous pericarditis [17]. In the United States, tuberculosis is also the leading cause of HIV-associated pericardial effusion, but numbers as divergent as 0%–15% have been reported for prevalence [18]. Further diagnostic workup clearly should be performed in developed countries.

A positive tuberculin skin test result may increase the suspicion of tuberculous pericarditis, but a negative skin test result does not exclude this diagnosis. In our series, a tuberculin skin test was performed in 7 cases, and 4 (57%) of the patients had an anergic response to both tuberculin and the control antigen. Of the 3 patients with positive purified protein derivatives (PPDs) of tuberculin skin tests, 1 had been PPD positive 13
<table>
<thead>
<tr>
<th>Subsequent procedures</th>
<th>Pericardial tissue pathology</th>
<th>Steroids given?</th>
<th>Culture results</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Granulomas</td>
<td>No</td>
<td>Tissue grew <em>Mycobacterium tuberculosis</em> at 9 days, fluid at 16 days (after discharge), and sputum at 21 days</td>
<td>No cardiac symptoms 3 months later, elevated liver enzymes on antituberculous medications</td>
</tr>
<tr>
<td>Partial pericardiectomy</td>
<td>Granulomas and AFB</td>
<td>Yes</td>
<td>Fluid and tissue cultures negative at 2 weeks</td>
<td>Echocardiogram 1 month later suggestive of constrictive pericarditis</td>
</tr>
<tr>
<td></td>
<td>Granulomas</td>
<td>Yes</td>
<td>Sputum with AFB, pericardial fluid negative, no tissue cultured</td>
<td>Diagnosed with lung adenocarcinoma; died 6 weeks later</td>
</tr>
<tr>
<td>Repeat pericardiocentesis pericardial window</td>
<td>Fibrosis, chronic inflammation, AFB</td>
<td>Yes</td>
<td>Fluid and tissue cultures negative, had been on INH 15 days. PCR was positive in 1/3 fluid specimens.</td>
<td>Symptoms and electrocardiogram changes of pericarditis 2 years later</td>
</tr>
<tr>
<td>Repeat pericardiocentesis pericardial window</td>
<td>Chronic inflammation, granulomas</td>
<td>No</td>
<td>Pericardial fluid grew <em>M. tuberculosis</em> after discharge, no tissue cultured</td>
<td>Discharged home at 2 weeks</td>
</tr>
<tr>
<td>Pericardiectomy for constrictive pericarditis</td>
<td>Granulomas</td>
<td>Yes (2 months later)</td>
<td>Pericardial fluid grew <em>M. tuberculosis</em>, tissue sent after 2 months on therapy was negative</td>
<td>Constrictive pericarditis</td>
</tr>
<tr>
<td></td>
<td>Not sent</td>
<td>No</td>
<td>Pericardial fluid grew &lt;10 colonies <em>M. tuberculosis</em> after discharge, no tissue cultured</td>
<td>Received 11 months directly observed therapy, no cardiac symptoms at 4.5 years</td>
</tr>
<tr>
<td>Pericardial window</td>
<td>Granulomas</td>
<td>Yes</td>
<td>Fluid grew <em>M. tuberculosis</em> after discharge, no tissue cultured</td>
<td>Took antituberculous medications for 6 months, no cardiac symptoms at 10 months</td>
</tr>
<tr>
<td>Pericardial window</td>
<td>Granulomas and AFB</td>
<td>No</td>
<td>No cultures sent</td>
<td>Discharged after 6 weeks</td>
</tr>
</tbody>
</table>

years prior to his admission for tuberculous pericarditis; this patient was not retested, so his PPD status at the time of admission was not known. Case series from before the HIV era document a negative PPD in ≤50% of proven cases of tuberculous pericarditis [2, 6, 19, 20]. The stage of disease at the time of presentation may affect the response to tuberculin testing, with patients presenting later in the course of illness being less likely to respond [15].

**Diagnosis: the importance of pericardial tissue.** Although physicians generally appreciate the need to obtain tissue samples, rather than fluid samples alone, for the diagnosis of tuberculous pleuritis, the same level of understanding may not extend to tuberculous pericarditis. Pericardial fluid culture alone is neither a reliable nor a timely method of making a diagnosis. In our case series, pericardial fluid was sent for culture in 9 cases, and in 6 (67%) of these cases, it eventually became positive. In 5 (83%) of 6 cases, however, the patient had already been discharged from the hospital. In 1 case, the fluid did not turn positive for AFB until 28 days after surgery, long after the patient had been transferred, untreated, to another hospital.

A predominant theme in our case series is the failure to submit pericardial tissue for culture before antituberculous therapy was given. In 6 (60%) of 10 cases, pericardial tissue specimens were not sent for culture, and, in 2 of 4 cases in which it was cultured, the patient had been taking isoniazid for >2 weeks. Overall, only 2 of 10 patients had pericardial tissue cultured before starting isoniazid, and 1 of these 2 cultures yielded *M. tuberculosis*. In the 1 instance in which fluid and tissue were both positive, the tissue culture was positive 5 days earlier than the fluid culture.

In most cases, antituberculous therapy was begun after gran-
ulomas were seen on histologic examination of pericardial tissue. Pericardial tissue was sent for biopsy in 13 cases, and all biopsy specimens revealed either granulomas or AFB. These cases suggest that the optimal diagnostic workup for tuberculous pericarditis includes pericardial window with tissue sent for both culture and histopathological examination before antituberculous medications are started.

Reviewing previous series of tuberculous pericarditis gives further insight into the difficulty of establishing the diagnosis. Fowler and Manitas [2] reported 19 cases of tuberculous pericarditis diagnosed between 1948 and 1972 in Cincinnati. Pericardial fluid was obtained from 13 patients, and AFB were seen on fluid staining in 5 of 12. In 10 of 13 cases, AFB were recovered on stained smear, culture, or guinea pig inoculation. The remaining 3 cases were diagnosed by means of pericardial tissue culture or histopathological examination. Rooney et al. [6] studied 35 patients with tuberculous pericarditis in New York in the 1960s. Fluid culture yielded M. tuberculosis in only 50% of the cases. Pericardial tissue established the diagnosis in 10 of 12 cases. In a series from 1960–1976 of 41 British patients with acute tuberculous pericarditis, M. tuberculosis grew in only 4 of 13 cultures of pericardial fluid; no biopsies were done [3]. Seventeen patients in New York with tuberculous pericarditis in the 1980s had a positive pericardial tissue culture in 7 of 7 cases, a positive pericardial fluid culture in 6 of 7 cases, and a positive pericardial fluid smear for AFB in 0 of 7 cases [20].

Eight of 11 biopsy specimens provided histopathological evidence of tuberculosis. Percutaneous pericardial biopsy was performed on 19 Kuwaiti children and adolescents, 7 of whom had tuberculous pericarditis [21]. Histopathological examination revealed tuberculous granulomas in 7 of 7 cases. Five (71%) of 7 tissue specimens (versus 2 [29%] of 7 of fluid cultures) grew M. tuberculosis. As these series indicate, the results of pericardial fluid culture are frequently falsely negative, and pericardial biopsy has a higher yield of diagnostic specimens.

In an attempt to improve the yield and shorten the time of the diagnostic workup, recent studies have utilized molecular biology techniques. Cegielski et al. [22] compared the reliability of PCR-based testing, culture, and histopathological examination for diagnosis of tuberculous pericarditis. In their study, 36 pericardial fluid specimens and 19 pericardial tissue specimens were obtained from 20 Tanzanian patients with large pericardial effusions. Sixteen of these patients had tuberculous pericarditis diagnosed on the basis of either positive culture results or the finding of typical granulomas in biopsy tissue together with response to therapy and lack of alternative diagnosis. When data were analyzed in terms of patients, all 3 methods proved to be useful for diagnosing tuberculous pericarditis. The sensitivity and specificity of culture and histopathological examination were slightly higher than those for PCR-based testing (for culture, 94% and 100%; for histopathological examination, 87% and 100%; and for PCR, 81% and 75%, respectively). When the data were analyzed in terms of individual specimens rather than patients, however, the value of the pericardial tissue versus pericardial fluid becomes clear. Cultures of pericardial tissue were positive in 14 (93%) of 15 specimens, whereas cultures of fluid were positive in only 16 (57%) of 28 specimens (P = .02, by use of \( \chi^2 \) test). Only 2 (15%) of 13 specimens of pericardial fluid obtained from patients with tuberculous pericarditis tested positive by PCR-based testing, compared with 12 (80%) of 15 specimens of pericardial tissue. Although PCR-based testing provided results more rapidly than did culture, it gave a false-positive result in 1 of 4 samples (this single patient had staphylococcal pericarditis). Therefore, its role remains uncertain.

The data from Cegielski et al. [22] are derived from specimens obtained from Tanzanian patients. In Tanzania, tuberculosis is the most common etiology of pericardial effusion, but in the United States, the prevalence is much lower. The current prevalence is not known, but large series from Spain, Russia, the United States, and Israel from the era before HIV suggested that tuberculosis accounted for <10% of pericardial effusions [14]. The lower prevalence of this disease may reduce the positive predictive value of PCR, culture, and histopathological examination in nonendemic areas.

**Open surgical drainage versus pericardiocentesis.** Two issues arise in the treatment of tuberculous pericarditis: the use of corticosteroids and the need for open surgical drainage versus pericardiocentesis. The goal of therapy of tuberculous pericarditis is not only to treat the acute symptoms of tamponade, but also to prevent progression from the effusive to the constrictive stage, in which a fibrotic and calcified pericardium entraps the heart [8, 23]. Our cases highlight the importance of pursuing an open procedure, rather than pericardiocentesis, to achieve a sustained relief of symptoms. Six of 10 patients initially had pericardiocentesis. Of these patients, 5 (83%) required subsequent drainage procedures, window placement, or pericardietomy. The number of patients with initial pericardiocentesis who required repeat procedures for drainage of reaccumulated fluid is striking.

The most thorough study of pericardiocentesis versus open surgical drainage was performed in 240 South African patients with effusive tuberculous pericarditis between July 1980 and September 1984 [24]. After diagnostic pericardiocentesis, the patients who were willing to undergo surgery, if so assigned, were randomly allocated to open pericardial biopsy and complete surgical drainage of fluid or to repeated percutaneous pericardiocentesis, as needed, to control symptoms and signs. Within each group, patients were further randomized to receive, or not to receive, prednisolone. All patients were treated with isoniazid, streptomycin, rifampicin, and pyrazinamide. Of the 48 patients randomized to open drainage, 4 died of pericarditis,
including 1 from the procedure; none needed repeated pericardiocentesis (table 2). Of the 53 patients randomized to pericardiocentesis alone, 3 died of pericarditis, 12 required repeated pericardiocentesis, and 5 required subsequent open drainage. These outcomes suggest that patients with tuberculous pericarditis who undergo open drainage are less likely to require repeated pericardiocentesis at a later date than are those who initially undergo bedside pericardiocentesis. Improved operative techniques now make subxyphoid pericardiectomy with pericardial biopsy a safer procedure [25]. In areas where tuberculous pericarditis is relatively uncommon, the need to obtain a pericardial biopsy for diagnosis also strongly favors the open procedure.

Although the immediate goal in the management of tuberculous pericarditis is relief of symptoms, the long-term goal is prevention of progression to constrictive pericarditis. Whether early, open drainage reduces this risk is unclear. Data from historical series clearly indicate that constrictive pericarditis can occur despite treatment with antituberculous medications and corticosteroids [5, 6, 26]. In the South African study, there was a tendency for patients in the open drainage group to require later pericardiectomy for constrictive pericarditis less often than did those in the pericardiocentesis group (2 [4%] of 48 patients vs. 5 [9%] of 53 patients), but this difference was not statistically significant [24]. A series of 16 Thai patients found that the presence of tamponade at admission correlated closely with development of constrictive pericarditis within the next 12 months (7 of 8 patients vs. 2 of 8 patients) [26]. This and other references suggest that the outcome of pericardiectomy is closely related to the degree of preoperative disability [27–29]. Whether surgical techniques do not permit complete release of advanced constriction or whether myocardial atrophy accounts for the postoperative myocardial dysfunction is unclear.

**The role of corticosteroids.** Five of our 10 patients were treated with corticosteroids, including the patient who developed constrictive pericarditis. This patient was not treated until after he already had clear symptoms of cardiac decompensation. Steroids were not given to the 3 HIV-positive patients, most likely out of concern for their immune status. Because the length of follow-up in these cases was not adequate to determine how many patients ultimately developed constrictive pericarditis, it is difficult to draw any conclusions about the value of steroids from this series.

The best study of the corticosteroid issue is again the South African study [24]. Two of 76 patients, who were randomized to receive 60 mg of prednisolone for first 4 weeks of treatment, followed by a tapering dose during the next 7 weeks, died of pericarditis, compared with 10 of 74 patients in the placebo group ($P < .05$; table 2). Nine percent of the steroid-treated patients required repeated pericardiocentesis versus 23% of the placebo group ($P < .05$). There was a trend toward fewer emergent open drainages for tamponade in the corticosteroid-treated group, but this difference did not achieve statistical significance. The rate of progression to constriction requiring pericardiectomy also showed a trend favoring corticosteroids that was not significantly different between the 2 groups.

The same authors performed a parallel study of patients in Transkei, South Africa, who had already developed the constrictive stage of tuberculous pericarditis [30]. The results were similar in that steroids shortened the time to resolution of symptoms, such as tachycardia and restriction of physical activity, although no significant differences were noted in the need for pericardiectomy or death because of pericarditis. Thus, in neither study did steroids significantly affect the risk of death or the progression to chronic constrictive pericarditis. At the present time, the potential benefit of corticosteroids is to quicken resolution of symptoms and decrease reaccumulation of fluid [31]. If used, steroids should be given before irreversible constriction has occurred [30–32]. No studies have specifically addressed the use of corticosteroids in HIV-positive patients with tuberculous pericarditis [15, 32].

**HIV and tuberculous pericarditis.** The association between HIV and pericardial disease is of sufficient importance to merit special mention. In Tanzania in the 1980s, 28 (72%) of 39 patients with a large pericardial effusion had HIV infection; the researchers postulated that tuberculosis was the common link [33]. In the developed world, the incidence of HIV-associated pericardial effusion is lower but still significant. A literature review of English-language journals from North America and Europe from 1982 to 1996 revealed that, in 1139 HIV-infected patients, the average incidence of pericardial disease of all kinds was 21% (the percentages were 9%–32% by autopsy and 8%–53% by echocardiogram assessment) [34]. Most effusions were small to moderate and asymptomatic. These small effusions may be a direct consequence of HIV infection with invasion of cardiac tissue by the virus itself [35].

### Table 2. Summary of reported results of corticosteroids and open surgical intervention on the outcome of tuberculous pericarditis [24].

<table>
<thead>
<tr>
<th>Adverse events</th>
<th>Steroid given</th>
<th>Open surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes ($n = 76$)</td>
<td>No ($n = 74$)</td>
</tr>
<tr>
<td>Death from pericarditis</td>
<td>2 (3)</td>
<td>10 (14) $^b$</td>
</tr>
<tr>
<td>Pericardiectomy</td>
<td>6 (8)</td>
<td>9 (12)</td>
</tr>
<tr>
<td>Repeated pericardiocentesis</td>
<td>7 (9)</td>
<td>17 (23) $^b$</td>
</tr>
<tr>
<td>Subsequent open drainage</td>
<td>3 (4)</td>
<td>7 (9)</td>
</tr>
</tbody>
</table>

$^a$ At 24 months after the administration of corticosteroids or surgery.

$^b$ P < .05.

$^c$ P < .01.
In the small number of cases in which the effusion progresses to tamponade, however, tuberculosis accounts for 25% of the cases [34, 36, 37]. It is worth noting that 3 of our 10 patients had HIV infection.

The effect of HIV on the response to treatment of pericardial tuberculosis has not been well characterized [38]. In our series, HIV-positive patients did not differ from HIV-negative patients with regard to the short-term outcome of tuberculous pericarditis. All 3 HIV-positive patients responded well to antituberculous medications, although the follow-up time was relatively short. Cegielski et al. [14] found that 14 HIV-infected Tanzanian patients with tuberculous pericarditis responded as well as HIV-negative patients with tuberculous pericarditis did to antituberculous therapy. They believed that these patients were at an early stage of HIV infection, because 13 of 14 had strongly positive tuberculin skin test results; CD4 cell counts were not measured. Other case reports from Europe and the United States have likewise documented a good response to antituberculous therapy in HIV-positive patients with tuberculous pericarditis [39, 40]. Some authors have suggested that HIV does not impair the initial response to tuberculous therapy but is associated with a higher risk of relapse, or possibly re-infection [38, 41]. Our patient’s sputum samples did yield M. tuberculosis, although the patient lacked clinical signs of pulmonary tuberculosis. A study of 61 patients in Zimbabwe with tuberculous pericarditis found that the HIV-positive patients were more likely than HIV-negative patients to have disseminated tuberculosis; perhaps dissemination worsens the long-term outcome [15].

CONCLUSIONS

Tuberculosis is a relatively common cause of clinically significant pericarditis in HIV-positive patients. Pericardial tissue specimens should be obtained to provide the best chance of definitive diagnosis. In cases characterized by large pericardial effusions, an open pericardial biopsy procedure is useful not only for diagnostic purposes but also to help prevent reaccumulation of fluid. Data on whether to use corticosteroids in the management of this disease in the HIV population are not available, but in the population without HIV, corticosteroids probably aid hemodynamic recovery. Overall, the information available on tuberculous pericarditis is sparse, and major texts tend to quote the same articles from African studies. Cumulative experience from published case studies in the developed world should contribute to better understanding of this disease.

References

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